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COVER ARTICLE

# Management of Peripheral Arterial Disease

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**Peripheral arterial disease** is common, but the diagnosis frequently is overlooked because of subtle physical findings and lack of classic symptoms. Screening based on the ankle brachial index using Doppler ultrasonography may be more useful than physical examination alone. Noninvasive modalities to locate lesions include magnetic resonance angiography, duplex scanning, and hemodynamic localization. Major risk factors for **peripheral arterial disease** are cigarette smoking, diabetes mellitus, older age (older than 40 years), hypertension, hyperlipidemia, and hyperhomocystinemia. Nonsurgical therapy for intermittent claudication involves risk-factor modification, exercise, and pharmacologic therapy. Based on available evidence, a supervised exercise program is the most effective treatment. All patients with **peripheral arterial disease** should undergo aggressive control of blood pressure, sugar intake, and lipid levels. All available strategies to help patients quit smoking, such as counseling and nicotine replacement, should be used. Effective drug therapies for **peripheral arterial disease** include aspirin (with or without dipyridamole), clopidogrel, cilostazol, and pentoxifylline. (Am Fam Physician 2004;69:525-32,533. Copyright© 2004 American Academy of Family Physicians.)

A patient information handout on **peripheral arterial disease**, provided by an AAFP staff patient education writer, is provided on page 533.

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See page 465

Although **peripheral arterial disease** (PAD) affects approximately 12 million persons in the United States, a recent study[1] con-cluded that many physicians routinely do not obtain a relevant history for PAD and frequently overlook subtle signs of the condition on physical examination *(Tables 1 and 2)*. The underdiagnosis of PAD in primary care may thwart effective secondary preventive strat-egies,[2] including intensive treatment for hyperlipidemia, hypertension, and smoking cessation. [Evidence level C, descriptive study]

**for definitions of strength-of-evidence levels.**

TABLE 1
**Differential Diagnosis of Intermittent Claudication**

| |
|---|
| **Nonvascular causes** |
| Arthritis of the hips |
| Restless legs syndrome |
| **Peripheral** neuropathies |
| Spinal stenosis (pseudoclaudication) |
| Prolapsed intervertebral disc |
| **Vascular causes** |
| **Arterial** embolus |
| Thromboangiitis obliterans (Buerger's **disease**) |
| Deep venous thrombosis |

TABLE 2
**Important Signs of Chronic Arterial Insufficiency**

| |
|---|
| Limb examination (and comparison with the opposite limb) includes: |
| Hair loss |
| Poor nail growth (brittle nails) |
| Dry, scaly, atrophic skin |
| Dependent rubor |
| Pallor with leg elevation after one minute at 60 degrees (normal color should return in 10 to 15 seconds; longer than 40 seconds indicates severe ischemia) |
| Ischemic tissue ulceration (punched-out, painful, with little bleeding), gangrene |
| Absent or diminished femoral or pedal pulses (especially after exercising the limb) |
| **Arterial** bruits |
| Additional examination by palpation and auscultation to detect abnormal aortic aneurysm or bruit |

## Diagnosis

Screening based on the ankle brachial index (ABI) measured by Doppler ultrasonography could prove highly useful in identifying patients with previously unrecognized PAD.[2] In a re-cent multicenter study,[3] the ABI correlated more closely with exercise capacity than did symptoms. This finding implies that many patients with PAD may not have the classic symptoms of claudication.[3] Some experts argue that a thorough physical examination with special attention to the pulses, auscultation for **arterial** bruits, and inspection for postural color changes *(Figure 1)* can be almost as informative as an ABI using Doppler ultrasonography.[4]

Several factors complicate the diagnosis of PAD. A normal ABI does not exclude a proximal aneurysm or **arterial** occlusive **disease** distal to the ankle.[4] Obtaining a medical history also can be problematic.[1,5]

Although 83 percent of the patients in one large study[2] knew they had PAD, only 49 percent of their physicians were aware of this history. More than one half of patients identified as having PAD on the basis of an abnormal ABI value do not have typical claudication symptoms, but they do have other types of leg pain on exertion, with reduced ambulatory activity and quality of life.[3]

**FIGURE 1.** Dermatologic findings of **peripheral arterial** occlusive **disease**.

Even advanced PAD may not produce claudication or other symptoms if the occlusion develops slowly, allowing sufficient collateral circulation to develop, or if the patient is mostly sedentary.[4] Improving skills in eliciting symptoms, ex-amining the **peripheral** vascular system, and obtaining segmental blood pressures *(Figure 2)*,[6] plus increased use of Doppler ABI in patients at risk of PAD, should identify more patients in whom aggressive preventive strategies might delay **disease** progression or obviate the need for an invasive intervention.[1,2]

## Treatment

Medical therapy for intermittent claudication involves risk-factor modification, exercise training, and pharmacologic therapy *(Figure 3)*.

Exercise training is the most effective treatment of **peripheral arterial disease**.

### RISK-FACTOR MODIFICATION

Cigarette smoking, diabetes mellitus, hy-pertension, hyperlipidemia, age older than 40 years, and hyperhomocystinemia increase the risk of developing PAD. All patients with PAD, regardless of the severity of symptoms, should undergo risk-factor modification.

*Smoking.* Smoking is the most important risk factor and is correlated more closely with developing PAD than any other risk factor.[7] Smoking cessation probably reduces the severity of claudication; however a meta-analysis[8] concluded that it did not improve maximal treadmill walking distance. [Evidence level B, observational study] Cessation of cigarette smoking reduces the progression of **disease**, as shown by lower rates of amputation and lower incidences of rest ischemia in patients who quit, and it reduces the risks of myocardial infarction and death from other vascular causes.[8]

Currently, almost one fourth of adults in the United States smoke cigarettes, and 70 per-cent of smokers report that they want to quit.[9] Approximately one third of smokers try to stop smoking each year, but only 20 percent seek professional help. Fewer than 10 percent of smokers who attempt to quit on their own are successful over the long term.[9] Two approaches have strong evidence of efficacy for smoking cessation: pharmacotherapy and counseling.[9-11] Each is effective by itself, but the two combined achieve the highest rates of smoking cessation.[9,11] Clinical trials have demonstrated that a physician's advice to stop smoking increases the rates of smoking cessation in patients by approximately 30 percent.[12] Providing a brief three-minute counseling session is more effective than advising the patient to quit, and it doubles the cessation rate compared with no intervention.[12] Too often, physicians miss this critical opportunity.[11]

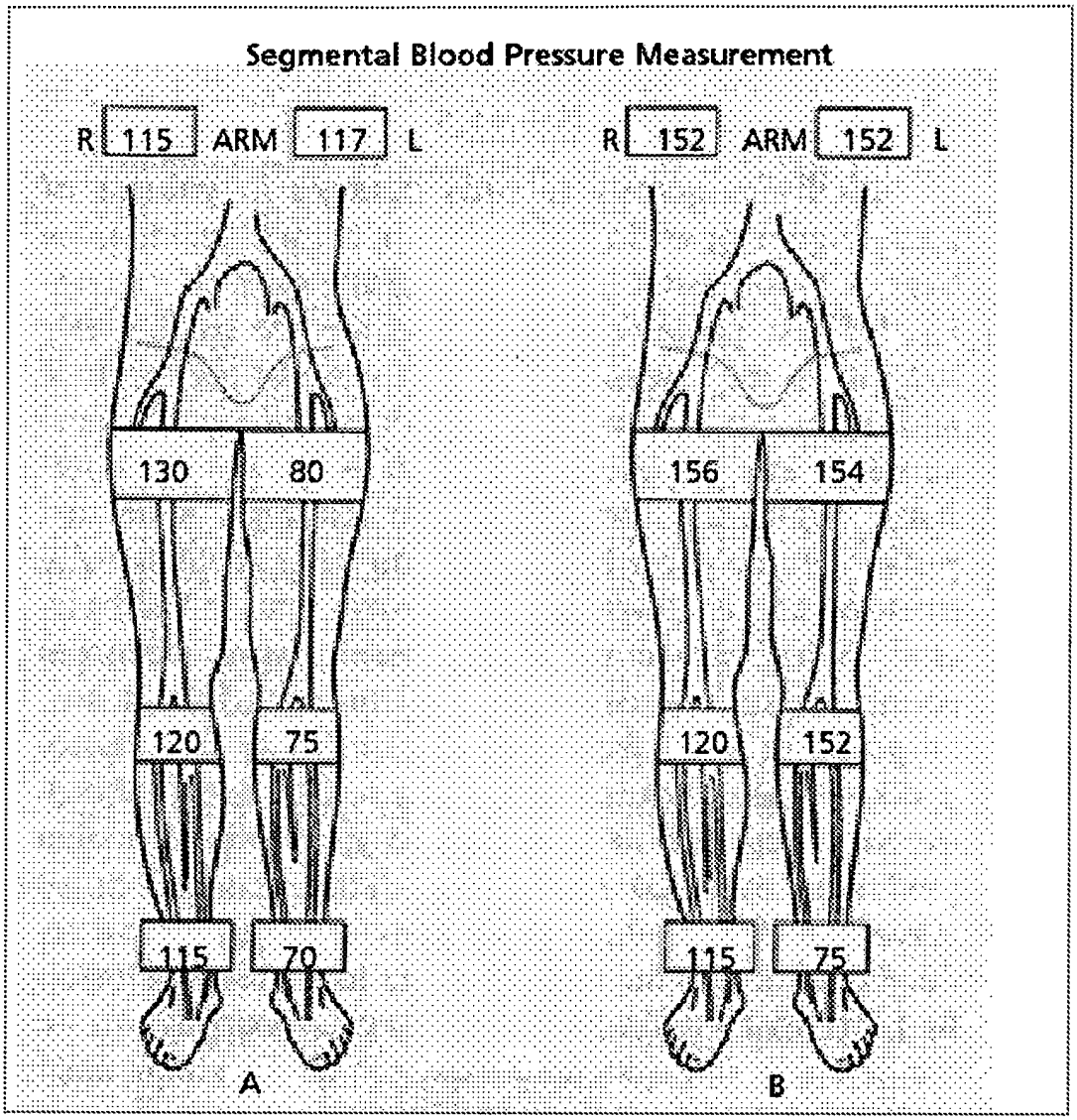


**FIGURE 2.** Segmental blood pressure measurement. A, segmental leg pressures in a normal right extremity (ABI: 115/115 = 1.00) and one with an isolated left iliac artery occlusion (ABI: 70/117 = 0.60). Horizontal and vertical pressure gradients exist at the thigh. B, segmental leg pressures in a patient with an isolated focal right superficial femoral artery stenosis and a distal left tibial artery occlusion. (ABI = ankle brachial index)

Adapted with permission from Wilt TJ. Current strategies in the diagnosis and management of lower extremity **peripheral** vascular **disease**. J Gen Intern Med 1992;7:91.

The U.S. Food and Drug Administration (FDA) has approved six products for smoking cessation: sustained-release bupropion (Zy-ban) and five nicotine-replacement products (i.e., gum, lozenge, a transdermal patch, a nasal spray, and a vapor inhaler). The use of all nicotine-replacement products increases the long-term rates of smoking cessation and relieves cravings for nicotine and the symptoms of nicotine withdrawal. Nortriptyline (Pamelor) and clonidine (Catapres) also have been found to aid smoking cessation, but the FDA has not approved them for this indication.

*Diabetes Mellitus.* No controlled trials have directly evaluated the effects of antidiabetic therapy on the natural history of PAD. Currently, no prospective evidence shows that tight glycemic control decreases the incidence of intermittent claudication or critical limb ischemia.[13] However, minimizing hyperglycemia as a risk factor associated with the subsequent development of PAD could not only decrease the rates of cardiovascular **disease** and myocardial infarction, but also reduce the occurrence of PAD and important PAD outcomes (claudication, **peripheral** revascularization, or critical limb ischemia and

amputation), as shown in the United Kingdom Prospective Diabetes Study (UKPDS 59).[14]

Intensive insulin therapy elicited a trend for reduced risk of important PAD outcomes (claudication, **peripheral** revascularization, or amputation) by 22 percent. This result did not achieve statistical significance, because the study was not powered for assessment of this outcome. In other words, there is at least moderately strong, if statistically inconclusive, evidence that macrovascular coronary (and potentially limb) outcomes are improved with glycemic control, and these outcomes are central to good PAD care. Even in the absence of high-quality clinical investigations, it is important to note that diabetic control has an impact on limb infection and amputation in patients with severe PAD (critical limb ischemia). Furthermore, because aggressive control of blood glucose in type 1 and type 2 diabetes reduces the risk of microvascular complications, it also may benefit patients with PAD.[13,14]

*Hypertension.* Hypertension is a major risk factor for PAD, but the effect of antihypertensive therapy on the progression of **disease** or the risk of claudication is unclear. Data derived from studies of cardiovascular **disease** support the aggressive treatment of hypertension in patients with PAD.[7] Although no data demonstrate an impact of antihypertensive therapy on PAD outcomes, this lack of data is because PAD-related event rates are low. The power to detect such outcomes would require a trial larger than the recent Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) effort, and a trial of efficacy would ethically require an active comparator intervention, making the assessment of PAD-specific and drug-specific outcomes essentially impossible.

Hypertension should be controlled in these patients primarily to reduce morbidity from cardiovascular and cerebrovascular **disease**. The use of beta blockers in patients with intermittent claudication was of concern because several early case reports documented worsening claudication and decreasing blood flow in the legs of patients taking these drugs. A later meta-analysis and critical review concluded that beta-adrenergic antagonists are safe in patients with PAD, except in those most severely affected. Even in these patients, the drugs can be administered, but with caution.[15,16]

National guidelines currently recommend aspirin (with or without dipyridamole) or clopidogrel as first-choice drugs for **peripheral arterial disease**.

*Hyperlipidemia.* Several large clinical trials have demonstrated the benefits of lipid-lowering therapy in patients with PAD who have coexisting coronary and cerebral **arterial disease**.[17-25] Simvastatin (Zocor) drastically lowered cardiovascular ischemic event rates in the large PAD subgroup, despite initial low low-density lipoprotein (LDL) levels.[25] Lipid normalization has been shown to reduce **disease** progression and the severity of claudication.[17-25] The current recommendation for patients is to achieve a serum LDL cholesterol concentration of less than 100 mg per dL (2.6 mmol per L) and a serum triglyceride concentration of less than 150 mg per dL (3.9 mmol per L).[18] A statin drug should be given as initial therapy, but niacin also is a consideration because it increases serum high-density lipoprotein (HDL) concentrations and lowers serum triglyceride concentrations without (as formerly believed) worsening glucose metabolism in susceptible patients.[19]

*Hyperhomocystinemia.* A high serum homo-cysteine concentration is an independent risk factor for PAD and is associated with an increased risk of death from cardiovascular causes.[26] The causes of high serum homocysteine concentrations include genetic defects in homocysteine metabolism, alterations in vitamin $B_{12}$ metabolism, and dietary folate deficiency. Although supplementing the diet with B vitamins and folate usually lowers serum homocysteine concentrations,[27] no controlled trials demonstrate that reducing serum homocysteine concentration is beneficial in patients with PAD.[28] PAD is not a contraindication to estrogen therapy, but estrogen should not be recommended for treating PAD in postmenopausal women.[28,29]

**THERAPY**

*Exercise.* A formal exercise program is the most effective treatment of PAD, and effectiveness was demonstrated in more than 20 controlled trials.[30-33] Exercise increased the distance to onset of claudication by 179 percent in a meta-analysis of 21 published studies.[33] The greatest improvements in walking ability occur when each exercise session lasts longer than 30 minutes, when sessions take place at least three times per week, when the patient walks until near-maximal pain is reached in each session, and when the program lasts at least six months.[33] These improvements were sustained when patients continued to participate in a maintenance program for an additional 12 months.[33]

Another meta-analysis[34] from the Cochrane Collaboration that considered only randomized controlled trials showed that exercise produced significant improvements in walking time, compared with angioplasty and anti-platelet therapy. Motivated patients in a supervised setting modeled after cardiac rehabilitation had the best results.[33] The large, exercise-induced improvements in function and symptoms that occur in patients with claudication do not appear to be caused by an increased collateral blood flow but by other mechanisms.[33] Such potential mechanisms include improvements in endothelial vaso-dilator function, inflammatory responses, skeletal-muscle metabolism, enhanced oxygen availability by improved blood viscosity, and lessened ischemia at any achieved workload.[33]

The recent publication of a current procedural terminology (CPT) code for PAD exercise reflects the importance of exercise therapy for PAD (CPT 93668; Current Procedural Terminology, American Medical Association, Chicago, Ill., 2001).

*Pharmacologic Therapy.* Although a meta-analysis of randomized studies of antiplatelet agents found that ticlopidine (Ticlid) had the best evidence of efficacy in improvement in walking distance and reduction in occlusion,[7,28] it is no longer used because it has been associated with life-threatening hematologic reactions and because of the availability of newer safer agents *(Table 3).* Aspirin and dipyridamole (Persantine) increase the pain-free walking distance and resting limb blood flow, or lead to an improved coagulation profile and ABI.[7,28,35]

TABLE 3
**Pharmacotherapy for Patients with Claudication**

| *Drugs* | *Dosage* | *Comments* |
|---|---|---|
| Aspirin | 81 to 325 mg per day orally | Recommended by the American College of Chest Physicians for PAD, but the FDA found insufficient evidence to approve labeling for this indication |
| Clopidogrel (Plavix) | 75 mg per day orally | Fewer side effects than aspirin in the CAPRIE trial; significantly less risk for TTP than ticlopidine |
| Pentoxifylline (Trental) | 1.2 g per day orally | May have a small effect on walking ability, but insufficient data to support widespread use |
| Cilostazol (Pletal) | 100 mg twice per day orally | Correct dosing is critical; avoid in patients with heart failure; reduce dosing to 50 mg twice per day in patients taking calcium channel blockers; may cause loose stools and gastric upset |
| Ticlopidine (Ticlid) | 500 mg per day orally | Extensive hemodynamic monitoring for risk of TTP |

| *Experimental therapies* | *Dosage* | *Comments* |
|---|---|---|
| Naftidrofuryl (Nafronyl) | 600 mg per day orally | Serotonin antagonist; increased walking distance in several trials, but use remains controversial; approved in Europe |
| Propionyl levocarnitine | 2 g per day orally | No significant evidence |
| Prostaglandins (beraprost, iloprost, prostaglandin $E_1$ [Alprostadil]) | 120 mcg per day orally, or 60 mcg per day parenterally | Inconsistent results in recent trials |
| Gene-induced angiogenesis with endothelial growth factor | | Promising results in uncontrolled trials |
| Ginkgo biloba extract | | Effective, but study methodology questionable |
| Hyperbaric oxygen | | Expensive; results equivocal |

PAD = **peripheral arterial disease**; FDA = U.S. Food and Drug Administration; CAPRIE = Clopidogrel versus Aspirin in Patients at Risk of Ischemic Events; TTP = thrombotic thrombocytopenic purpura.

In the Clopidogrel versus Aspirin in Patients at Risk of Ischemic Events (CAPRIE) trial,[36] 75 mg of clopidogrel was slightly but significantly better than 325 mg of aspirin in the total population for preventing strokes, myocardial infarction, and vascular **disease**.[36] The PAD subgroup with nearly 6,500 patients in CAPRIE was larger than any other prospective PAD clinical trial and demonstrated a profound superiority in the PAD population treated with clopidogrel compared with aspirin alone.[36] This result led to the FDA approval of clopidogrel (Plavix) for the secondary prevention of atherosclerotic events in patients with PAD.

Pentoxifylline (Trental), a rheologic modifier that also has an antiplatelet effect, was approved in 1984 for the treatment of claudication. Pentoxifylline is less effective than cilostazol (Pletal).[28,35] Two meta-analyses and two systematic reviews of pentoxifylline concluded that although the drug may have a small effect on walking ability, the data are insufficient to support its widespread use.[28,35] In a more recent controlled trial,[36] pentoxifylline was significantly superior to placebo in improving walking distance after six and 12 months of therapy.[37]

Cilostazol inhibits phosphodiesterase 3, sup-presses platelet aggregation, activates lipoprotein lipase, and causes **arterial** dilation.[7,28,35] Approved in 1999 by the FDA for the treatment of claudication, it improved pain-free and maximal treadmill walking distance in randomized controlled trials compared with placebo or pentoxifyl-line.[7,28,35] Correct dosing is important, because 100 mg orally twice per day significantly improved claudication symptoms, while 100 mg per day did not.[38] Cilostazol should not be used in patients with heart failure.[7,28] The dosage should be reduced to 50 mg orally twice per day when calcium channel blockers are being used because serum drug levels are elevated in these patients. Common side effects of cilostazol include loose stool and gastric upset.[7,28,35]

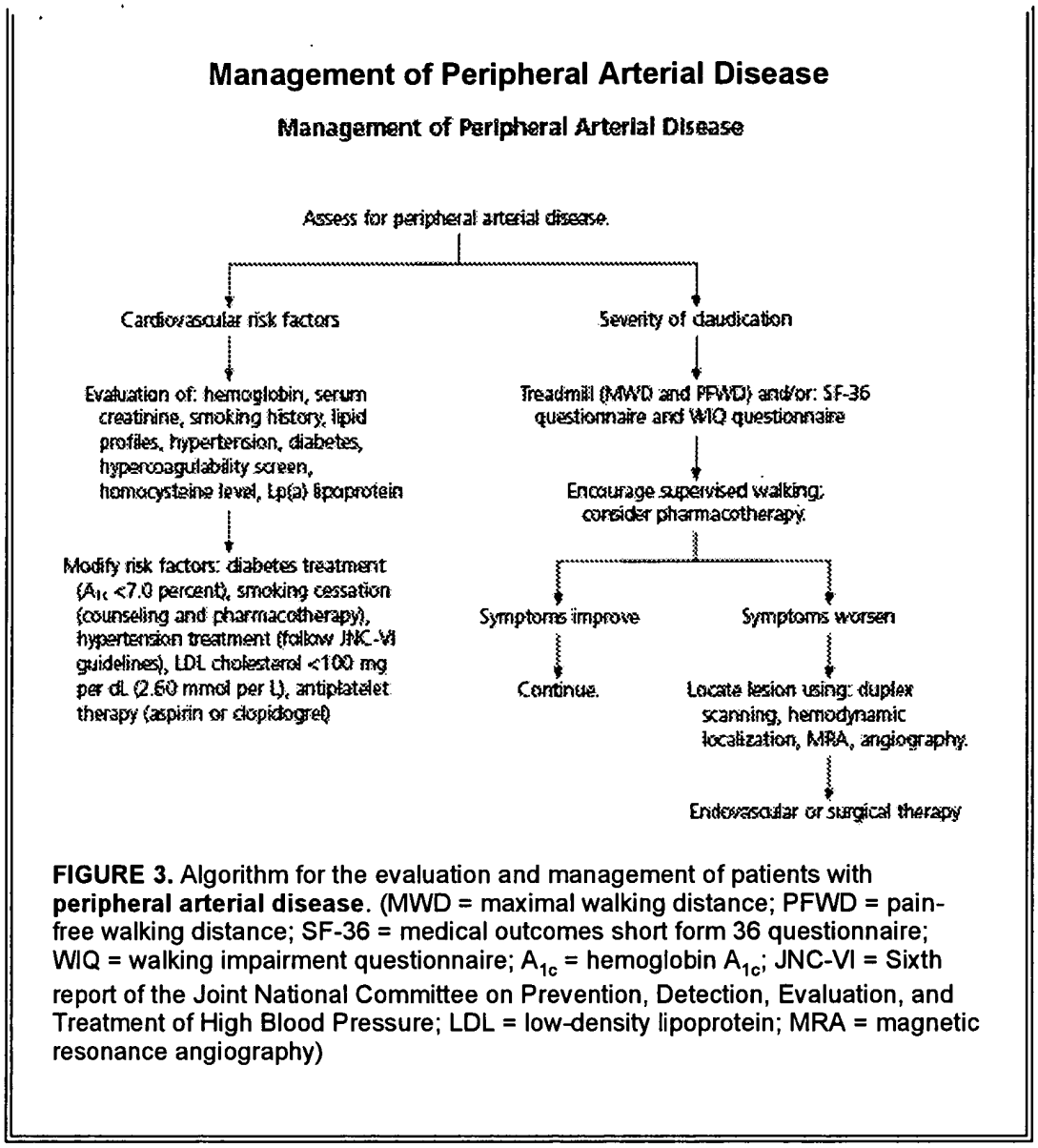


**FIGURE 3.** Algorithm for the evaluation and management of patients with **peripheral arterial disease.** (MWD = maximal walking distance; PFWD = pain-free walking distance; SF-36 = medical outcomes short form 36 questionnaire; WIQ = walking impairment questionnaire; $A_{1c}$ = hemoglobin $A_{1c}$; JNC-VI = Sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; LDL = low-density lipoprotein; MRA = magnetic resonance angiography)

In summary, aspirin generally is considered the antiplatelet drug of first choice. The 6th ACCP Consensus Conference recommends that aspirin alone (81 to 325 mg per day) or in combination with dipyridamole, should be given indefinitely because it can modify the natural history of intermittent claudication and those with high risk for future cardiovascular events.[39] These guidelines also suggest that clopidogrel may be superior to aspirin and should be considered as an alternative treatment in these patients.[39] Experimental or investigational agents for PAD include naftidrofuryl (Nafronyl), which is approved in Europe, macrolide antibiotic treatment for chlamydia infection, propionyl levocarnitine, defibrotide, ginkgo biloba, hyperbaric oxygen, and angiogenic growth factors. Of these, angiogenic growth factors are perhaps the most promising.[35] For an in-depth, evidence-based review of PAD management, physicians may refer to the Trans Atlantic Inter-Society Consensus (TASC).[40]

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# Peripheral Vascular Disease

**What is peripheral vascular disease?**

This refers to diseases of blood vessels outside the **heart** and brain. It's often a narrowing of vessels that carry blood to the legs, arms, stomach or kidneys. There are two types of these circulation disorders:

- **Functional** peripheral **vascular** diseases don't have an organic cause. They don't involve defects in blood vessels' structure. They're usually short-term effects related to "spasm" that may come and go. Raynaud's **disease** is an example. It can be triggered by cold temperatures, emotional stress, working with vibrating machinery or smoking.

- **Organic** peripheral **vascular** diseases are caused by structural changes in the blood vessels, such as inflammation and tissue damage. Peripheral artery **disease** is an example. It's caused by fatty buildups in arteries that block normal blood flow.

**What is peripheral artery disease?**

Peripheral artery **disease** (PAD) is a condition similar to coronary artery **disease** and carotid artery **disease**. In PAD, fatty deposits build up in the inner linings of the artery walls. These blockages restrict blood circulation, mainly in arteries leading to the kidneys, stomach, arms, legs and feet. In its early stages a common symptom is cramping or fatigue in the legs and buttocks during activity. Such cramping subsides when the person stands still. This is called "intermittent claudication." People with PAD often have fatty buildup in the arteries of the **heart** and brain. Because of this association, most people with PAD have a higher risk of death from **heart** attack and stroke.

**How is peripheral artery disease diagnosed and treated?**

Techniques used to diagnose PAD include a medical history, physical exam, ultrasound, X-ray angiography and magnetic resonance imaging angiography (MRA).

Most people with PAD can be treated with lifestyle changes, medications or both. Lifestyle changes to lower your risk include:

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DID YOU KNOW?

Of the 8-12 million people who are affected by peripheral artery **disease** (PAD) nearly 75% will never experience symptoms. Women are less likely to have symptoms than men.

read more...

- Stop smoking (smokers have a particularly strong risk of PAD).
- Control diabetes.
- Control blood pressure.
- Be physically active (including a supervised exercise program).
- Eat a low-saturated-fat, low-cholesterol diet.

PAD may require drug treatment, too. Drugs include:

- medicines to help improve walking distance (cilostazol and pentoxifylline).
- antiplatelet agents
- cholesterol-lowering agents (statins)

In a minority of patients, lifestyle modifications alone aren't sufficient. In these cases, angioplasty or surgery may be necessary.

Angioplasty is a non-surgical procedure that can be used to dilate (widen) narrowed or blocked peripheral arteries. A thin tube called a catheter with a deflated balloon on its tip is passed into the narrowed artery segment. Then the balloon is deflated and the catheter is withdrawn.

Often a stent — a cylindrical, wire mesh tube — is placed in the narrowed artery with a catheter. There the stent expands and locks open. It stays in that spot, keeping the diseased artery open.

If the narrowing involves a long portion of an artery, surgery may be necessary. A vein from another part of the body or a synthetic blood vessel is used. It's attached above and below the blocked area to detour blood around the blocked spot.

See the Related Items box above for links to the **Cardiology Patient Page** in *Circulation, Journal of the American **Heart** Association*:

- Diseases of the Veins

**Related AHA publications:**

- **Heart** and Stroke Facts
- **Heart Disease** and Stroke Statistics Update

**Related AHA Scientific Statements**
Peripheral **Vascular Disease**

**See also:**

Angioplasty and Cardiac Revascularization Treatments and Statistics
Angioplasty, Laser
Angioplasty, Percutaneous Transluminal Coronary (PTCA)
Anticoagulants
Atherectomy
Atherosclerosis
Bypass Surgery, Coronary Artery
Cardiac Catheterization
**Heart** Damage Detection
Raynaud's Phenomenon
Stent Procedure
Stroke Risk Factors
Stroke Tests
Thrombolysis, Thrombosis, Thrombus and Embolus

# Coronary heart disease

From Wikipedia, the free encyclopedia
(Redirected from Coronary artery disease)

It has been suggested that this article or section be merged with *Cardiovascular disease*. (Discuss)

**Coronary heart disease** (CHD), also called **coronary artery disease** (CAD) and atherosclerotic heart disease, is the end result of the accumulation of atheromatous plaques within the walls of the arteries that supply the myocardium (the muscle of the heart). While the symptoms and signs of coronary heart disease are noted in the advanced state of disease, most individuals with coronary heart disease show no evidence of disease for decades as the disease progresses before the first onset of symptoms, often a "sudden" heart attack, finally arise. After decades of progression, some of these atheromatous plaques may rupture and (along with the activation of the blood clotting system) start limiting blood flow to the heart muscle. The disease is the most common cause of sudden death.

**Coronary heart disease**

| | |
|---|---|
| **ICD-10** | I20-I25 |
| **ICD-9** | 410 (http://icd9.chrisendres.com/index.php?action=search&srchtext=410)-414 (http://icd9.chrisendres.com/index.php?action=search&srchtext=414), 429.2 (http://icd9.chrisendres.com/index.php?action=search&srchtext=429.2) |

## Contents



## Overview

Atherosclerotic heart disease can be thought of as a wide spectrum of disease of the heart. At one end of the spectrum is the asymptomatic individual with atheromatous streaks within the walls of the coronary arteries (the arteries of the heart). These streaks represent the early stage of atherosclerotic heart disease and do not obstruct the flow of blood. A coronary angiogram performed during this stage of disease may not show any evidence of coronary artery disease, because the lumen of the coronary artery has not decreased in caliber.

Over a period of many years, these streaks increase in thickness. While the atheromatous plaques initially expand into the walls of the arteries, eventually they will expand into the lumen of the vessel, affecting the flow of blood through the arteries. While it was originally believed that the growth of atheromatous plaques was a slow, gradual process, some recent evidence suggests that the gradual buildup of plaque may be complemented by small plaque ruptures which cause the sudden increase in the plaque burden due to accumulation of thrombus material.

Atheromatous plaques that cause obstruction of less than 70 percent of the diameter of the vessel rarely cause symptoms of obstructive coronary artery disease. As the plaques grow in thickness and obstruct more than 70 percent of the diameter of the vessel, the individual develops symptoms of obstructive coronary artery disease. At this stage of the disease process, the patient can be said to have ischemic heart disease. The symptoms of ischemic heart disease are often first noted during times of increased workload of the heart. For instance, the first symptoms include exertional angina or decreased exercise tolerance.

As the degree of coronary artery disease progresses, there may be near-complete obstruction of the lumen of the coronary artery, severely restricting the flow of oxygen-carrying blood to the myocardium. Individuals with this degree of coronary heart disease

typically have suffered from one or more myocardial infarctions (heart attacks), and may have signs and symptoms of chronic coronary ischemia, including symptoms of angina at rest and flash pulmonary edema.

A distinction should be made between myocardial ischemia and myocardial infarction. Ischemia means that the amount of oxygen supplied to the tissue is inadequate to supply the needs of the tissue. When the myocardium becomes ischemic, it does not function optimally. When large areas of the myocardium becomes ischemic, there can be impairment in the relaxation and contraction of the myocardium. If the blood flow to the tissue is improved, myocardial ischemia can be reversed. Infarction means that the tissue has undergone irreversible death due to lack of sufficient oxygen-rich blood.

An individual may develop a rupture of an atheromatous plaque at *any* stage of the spectrum of coronary heart disease. The acute rupture of a plaque may lead to an acute myocardial infarction (heart attack). It is unclear at present which plaques in an individual are more likely to rupture in the future and cause a heart attack.

# Pathophysiology

Limitation of blood flow to the heart causes ischemia (cell starvation secondary to a lack of oxygen) of the myocardial cells. When myocardial cells die from lack of oxygen, this is called a myocardial infarction (commonly called a heart attack). It leads to heart muscle damage, heart muscle death and later scarring without heart muscle regrowth.

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CHD is associated with smoking, obesity, hypertension and a chronic sub-clinical lack of vitamin C. A family history of CHD is one of the strongest predictors of CHD. Screening for CHD includes evaluating homocysteine levels, high-density and low-density lipoprotein (cholesterol) levels and triglyceride levels.

# Angina

The pain associated with very advanced CHD is known as angina, and usually presents as a sensation of pressure in the chest, arm pain, jaw pain, and other forms of discomfort. The word *discomfort* is preferred over the word *pain* for describing the sensation of angina, because it varies considerably among individuals in character and intensity and most people do not perceive angina as painful, unless it is severe. There is evidence that angina and CHD present differently in women and men.

Angina that occurs regularly with activity, upon awakening, or at other predictable times is termed stable angina and is associated with high grade narrowings of the heart arteries. The symptoms of angina are often treated with nitrate preparations such as nitroglycerin, which come in short-acting and long-acting forms, and may be administered transdermally, sublingually or orally. Many other more effective treatments, especially of the underlying atheromatous disease, have been developed.

Angina that changes in intensity, character or frequency is termed unstable. Unstable angina may precede myocardial infarction, and requires urgent medical attention. It is treated with oxygen, intravenous nitroglycerin, and morphine. Interventional procedures such as angioplasty may be done.

# Risk Factors

**The following are confirmed independent risk factors for the development of CAD, in order of decreasing importance:**

1. Hypercholesterolemia (specifically, serum LDL concentrations)
2. Smoking

3. Hypertension (high systolic pressure seems to be most significant in this regard)
4. Hyperglycemia (due to diabetes mellitus or otherwise)
5. Hereditary differences in such diverse aspects as lipoprotein structure and that of their associated receptors, homocysteine processing/metabolism, etc.

**Significant, but indirect risk factors include:**

- Male sex (by far the most significant of this group)
- Lack of exercise
- Stress
- Diet rich in saturated fats
- Obesity

# Prevention

Coronary heart disease is the most common form of heart disease in the Western world. Prevention centers on the modifiable risk factors, which include decreasing cholesterol levels, addressing obesity and hypertension, avoiding a sedentary lifestyle, making healthy dietary choices, and stopping smoking. There is some evidence that lowering uric acid and homocysteine levels may contribute. In diabetes mellitus, there is little evidence that blood sugar control actually improves cardiac risk. Some recommend a diet rich in omega-3 fatty acids and vitamin C.

An increasingly growing number of other physiological markers and homeostatic mechanisms are currently under scientific investigation. Among these markers are low density lipoprotein and asymmetric dimethylarginine. Patients with CHD and those trying to prevent CHD are advised to avoid fats that are readily oxidized (e.g., saturated fats and trans-fats), limit carbohydrates and processed sugars to reduce production of Low density lipoproteins while increasing High density lipoproteins, keeping blood pressure normal, exercise and stop smoking. These measures limit the progression of the disease. Recent studies have shown that dramatic reduction in LDL levels can cause mild regression of coronary heart disease.

Risk factor management is carried out during cardiac rehabilitation, a 4-phase process beginning in hospital after MI, angioplasty or heart surgery and continuing for a minimum of three months. Exercise is a main component of cardiac rehabilitation along with diet, smoking cessation and blood pressure and cholesterol management.

## Preventive diets

- Vegetarian diet: Vegetarians have been shown to have a 24% reduced risk of dying of heart disease (source: Key TJ, Fraser GE, et al. 1999, Sep. Mortality in vegetarians and nonvegetarians: detailed findings from a collaborative analysis of 5 prospective studies. Am J Clin Nutr, 70:516S-524S).

- Cretan Mediterranean-style diet: The Seven Country Study found that Cretan men had exceptionally low death rates from heart disease, despite moderate to high intake of fat. The Cretan diet is similar to other traditional Mediterranean diets: consisting mostly of olive oil, bread, abundant fruit and vegetables, a moderate amount of wine and a small amount of animal products. However, the Cretan diet consisted of less fish and wine consumption than some other Mediterranean-style diets, such as the diet in Corfu, another region of Greece, which had higher death rates.

# Recent research

*Further information: atheroma and atherosclerosis*

A 2006 study found a region on Chromosome 17 was confined to families with multiple cases of myocardial infarction[1] (http://genetics.plosjournals.org/perlserv/?request=get-document&doi=10%2E1371%2Fjournal%2Epgen%2E0020072#s0).

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for treatment and prevention of the disease beyond the many current, proven strategies.

Ornisch has suggested that coronary heart disease is partially reversible using an intense dietary regime such as the Cretan diet coupled with regular cardio exercise.

# External links

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Retrieved from "http://en.wikipedia.org/wiki/Coronary_heart_disease"

Categories: Articles to be merged | Articles with unsourced statements | Cardiovascular diseases | Ischemic heart disease

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## General Illness Information

**Common Name:** **Coronary Artery Disease**

**Medical Term:** *Ischemic Heart Disease*

**Description:** **Coronary artery disease** is a condition in which fatty deposits (atheroma) accumulate in the cells lining the wall of the **coronary** arteries. These fatty deposits build up gradually and irregularly in the large branches of the two main **coronary** arteries which encircle the **heart** and are the main source of its blood supply. This process is called atherosclerosis which leads to narrowing or hardening of the blood vessels supplying blood to the **heart** muscle (the **coronary** arteries ).This results in ischemia ( inability to provide adequate oxygen) to **heart** muscle and this can cause damage to the **heart** muscle . Complete occlusion of the blood vessel leads to a **heart** attack (myocardial infarction). In the United States , cardiovascular **disease** is the leading cause of death among both sexes, and **coronary artery disease** is the commonest cause of cardiovascular **disease**. Myocardial infarction causes 35% of deaths in men between 35 and 50.The death rate is higher for men than for women between the ages of 35 and 55.However, after the age 55, the death rate for men declines but the rate for women continues to climb.

**Causes:** Exact cause is unknown. However there are a number of risk factors. Control of these risk factors has been shown to reduce the severity and complications of the **disease**.

**Prevention:** It is now clear that reducing certain risk factors, we can both prevent **coronary artery disease** and delay its progression and complications after it it has become manifest. Treatment of lipid abnormalities has now been shown to delay the progression of atherosclerosis and in some cases has even produced regression of the atherosclerotic plaques

## Signs & Symptoms

- Early stages: No symptoms.

- Later stages:
  Angina pectoris (burning, squeezing, heaviness, or tightness in the chest that may extend to the left arm, neck, jaw, or shoulder blade). See Angina Pectoris.
  Typically, angina is precipitated by physical activity, lasting no more than a few minutes, and is relieved by rest. Usually angina is worse when exertion follows a meal. It is also worse in cold weather and can be triggered by walking from a warm room into the cold air. Emotional stress can also cause or worsen angina.

  Not all people with ischemia will present with angina. Ischemia without angina is called silent ischemia. It is not yet understood why ischemia is sometimes silent.

-

## Risk Factors

- ☐ Family history of **coronary artery disease**, diabetes, high blood pressure or atherosclerosis.
- ☐ Smoking.
- ☐ Poor nutrition, especially too much fat in the diet.
- ☐ Previous **heart** attack or stroke.
- ☐ Previous **heart** attack or stroke.
- ☐ Overweight
- ☐ Hypertension
- ☐ Elevated cholesterol and/or low level of HDL (high-density lipoprotein).
- ☐ Type A personality

## Diagnosis & Treatment

Diagnosis of angina is a *clinical diagnosis* based on a characteristic complaint of chest discomfort or chest pain brought on by exertion and relieved by rest. Confirmation may be obtained by observing reversible **ischemic** changes on ECG during an attack or by giving a test dose of sublingual nitroglycerin that characteristically relieves the pain in 1 to 3 minutes.

Certain tests may help determine the severity of ischemia and the presence and extent of the **coronary artery disease**. Diagnostic tests may include electrocardiogram (measures electrical activity of the **heart**), echocardiogram (measures sound waves), exercise-tolerance test, thallium stress test, blood studies to measure total fat, cholesterol and lipoproteins,

X-rays of the chest and **coronary** angiogram (cardiac catheterization).

**General Measures:**

- Stop smoking
- Treat elevated cholesterol levels with low fat, low cholesterol diet, exercise and cholesterol lowering medications
- Treat elevated blood pressure
- Reduce stress
- Maintain ideal body weight
- Balloon angioplasty (treatment for obstructed arteries, especially those supplying blood to the **heart** and brain. A small uninflated balloon is passed up the **artery** to the obstruction, and then expanded to release the obstruction
  Although these procedures may decrease or eliminate symptoms for a while, they do not control the underlying **disease**.
- Surgery to bypass **coronary** arteries (severe cases).
- End-stage **coronary artery disease**, even when no simple procedures will help, can still be cured with a **heart** transplant in rare cases.

**Medications:**

- Four types of medications are available: beta-blockers, nitrates, calcium channel antagonists and anti-platelet drugs.
- Beta-blockers- reduce the resting **heart** rate and so reduce the demand for oxygen. Beta-blockers and nitrates have been proven to reduce the incidence of **heart** attacks and sudden deaths in people with **coronary artery disease**.
- Nitrates-such as nitroglycerin, cause dilatation of the blood vessels. There are short-acting and long-acting nitrates . Nitroglycerin is available as a tablet (sublingual) or an oral spray. A tablet of nitroglycerin placed under the tongue or inhalation of the oral spray usually relieves an episode of angina in 1 to 3 minutes- the effect of these short-acting nitrates lasts 30 minutes. Anyone with chronic stable angina must keep nitroglycerin tablets or spray with them at all times.

  Long-acting nitrates are available as tablets, skin patches or paste. Tablets are taken 1 to 4 times daily. Nitro paste and skin patches, in which the drug is absorbed through the skin over many hours, are also effective. Long-acting nitrates do tend to lose their effectiveness when taken regularly and therefore it is recommended to have 8 to 12 hour interval without taking the drug to maintain its effectiveness.
- Calcium channel antagonists- prevent the blood vessels from constricting and thus prevent **coronary artery** spasm. Certain calcium antagonists, such as verapamil and diltiazem, also slow the **heart** rate and in some patients these drugs are used in conjunction with beta-blockers to prevent episodes of tachycardia (fast **heart** rate).
- Anti-platelet drugs- such as aspirin is recommended for patients with **coronary artery disease** . Aspirin binds irreversibly to platelets and prevents them from clumping on blood vessel walls- thus preventing platelets from forming a clot on the fatty plaques which could block an **artery** and result in **heart** attack.. Recommended dose is one baby aspirin or half an adult aspirin daily. For people with allergy to aspirin can be treated with .alternative medications such as ticlopidine or clopidogrel bisulphate

**Activity:**

Engage in a program of moderate, daily physical exercise. Resume sexual activity once medical permission is given.

**Diet:**

- Low-fat and low cholesterol diet.
- If you are overweight, begin a moderate reducing diet and stick to it.

**Possible Complications :**

- Angina pectoris
- Life-threatening myocardial infarction (death of **heart** muscle cells from inadequate blood supply).
- Sudden death

## Prognosis

Treatment can prolong life and improve its quality.
Tremendous amount of research in this field, and new advances are being made and increasing evidence that aggressive treatment can reverse or arrest course of this **disease**. It is very important to follow your doctor's instructions, especially with respect to lifestyle changes and cholesterol reduction.
Long term prognosis depends on a number of key factors such as the age, the extent of **coronary artery disease**, the severity of symptoms and most of all , the pumping ability of the **heart**.

## Other

'Nothing Specified'.

# Coronary heart disease

From Wikipedia, the free encyclopedia

It has been suggested that this article or section be merged with *Cardiovascular disease*. (Discuss)

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**Coronary heart disease**

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## Overview

Atherosclerotic **heart disease** can be thought of as a wide spectrum of **disease** of the **heart**. At one end of the spectrum is the asymptomatic individual with atheromatous streaks within the walls of the **coronary** arteries (the arteries of the **heart**). These streaks represent the early stage of atherosclerotic **heart disease** and do not obstruct the flow of blood. A **coronary** angiogram performed during this stage of **disease** may not show any evidence of **coronary artery disease**, because the lumen of the **coronary artery** has not decreased in caliber.

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originally believed that the growth of atheromatous plaques was a slow, gradual process, some recent evidence suggests that the gradual buildup of plaque may be complemented by small plaque ruptures which cause the sudden increase in the plaque burden due to accumulation of thrombus material.

Atheromatous plaques that cause obstruction of less than 70 percent of the diameter of the vessel rarely cause symptoms of obstructive **coronary artery disease**. As the plaques grow in thickness and obstruct more than 70 percent of the diameter of the vessel, the individual develops symptoms of obstructive **coronary artery disease**. At this stage of the **disease** process, the patient can be said to have **ischemic heart disease**. The symptoms of **ischemic heart disease** are often first noted during times of increased workload of the **heart**. For instance, the first symptoms include exertional angina or decreased exercise tolerance.

As the degree of **coronary artery disease** progresses, there may be near-complete obstruction of the lumen of the **coronary artery**, severely restricting the flow of oxygen-carrying blood to the myocardium. Individuals with this degree of **coronary heart disease** typically have suffered from one or more myocardial infarctions (**heart** attacks), and may have signs and symptoms of chronic **coronary** ischemia, including symptoms of angina at rest and flash pulmonary edema.

A distinction should be made between myocardial ischemia and myocardial infarction. Ischemia means that the amount of oxygen supplied to the tissue is inadequate to supply the needs of the tissue. When the myocardium becomes **ischemic**, it does not function optimally. When large areas of the myocardium becomes **ischemic**, there can be impairment in the relaxation and contraction of the myocardium. If the blood flow to the tissue is improved, myocardial ischemia can be reversed. Infarction means that the tissue has undergone irreversible death due to lack of sufficient oxygen-rich blood.

An individual may develop a rupture of an atheromatous plaque at *any* stage of the spectrum of **coronary heart disease**. The acute rupture of a plaque may lead to an acute myocardial infarction (**heart** attack). It is unclear at present which plaques in an individual are more likely to rupture in the future and cause a **heart** attack.

# Pathophysiology

Limitation of blood flow to the **heart** causes ischemia (cell starvation secondary to a lack of oxygen) of the myocardial cells. When myocardial cells die from lack of oxygen, this is called a myocardial infarction (commonly called a **heart** attack). It leads to **heart** muscle damage, **heart** muscle death and later scarring without **heart** muscle regrowth.

Myocardial infarction usually results from the sudden occlusion of a **coronary artery** when a plaque ruptures, activating the clotting system and atheroma-clot interaction fills the lumen of the **artery** to the point of sudden closure. The typical narrowing of the lumen of the **heart artery** before sudden closure is typically 20%, according to clinical research completed in the late 1990s and using IVUS examinations within 6 months prior to a **heart** attack. High grade stenoses as such exceeding 75% blockage, such as detected by stress testing, were found to be responsible for only 14% of acute **heart** attacks the rest being due to plaque rupture/ spasm. The events leading up to plaque rupture are only partially understood. Myocardial infarction is also caused, far less commonly, by spasm of the **artery** wall occluding the lumen, a condition also associated with atheromatous plaque and CHD.

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# Angina

The pain associated with very advanced CHD is known as angina, and usually presents as a sensation of pressure in the chest, arm pain, jaw pain, and other forms of discomfort. The word *discomfort* is preferred over the word *pain* for describing the sensation of angina, because it varies considerably among individuals in character and intensity and most people do not perceive angina as painful, unless it is severe. There is evidence that angina and CHD present differently in women and men.

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# Risk Factors

**The following are confirmed independent risk factors for the development of CAD, in order of decreasing importance:**

1. Hypercholesterolemia (specifically, serum LDL concentrations)
2. Smoking
3. Hypertension (high systolic pressure seems to be most significant in this regard)
4. Hyperglycemia (due to diabetes mellitus or otherwise)
5. Hereditary differences in such diverse aspects as lipoprotein structure and that of their associated receptors, homocysteine processing/metabolism, etc.

**Significant, but indirect risk factors include:**

- Male sex (by far the most significant of this group)
- Lack of exercise
- Stress
- Diet rich in saturated fats
- Obesity

# Prevention

**Coronary heart disease** is the most common form of **heart disease** in the Western world. Prevention centers on the modifiable risk factors, which include decreasing cholesterol levels, addressing obesity and hypertension, avoiding a sedentary lifestyle, making healthy dietary choices, and stopping smoking. There is some evidence that lowering uric acid and homocysteine levels may contribute. In diabetes mellitus, there is little evidence that blood sugar control actually improves cardiac risk. Some recommend a diet rich in omega-3 fatty acids and vitamin C.

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# Recent research

*Further information: atheroma and atherosclerosis*

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- Diabetes Complications: **Heart Disease**

Retrieved from "http://en.wikipedia.org/wiki/Coronary_heart_disease"

Categories: Articles to be merged | Cardiovascular diseases | **Ischemic heart disease**

# Ischaemic heart disease

From Wikipedia, the free encyclopedia
(Redirected from **Ischemic heart disease**)

**Ischaemic** (or **ischemic**) **heart disease** is a **disease** characterized by reduced blood supply to the **heart**. It is the most common cause of death in most western countries.

*Ischaemia* means a "reduced blood supply". The coronary arteries supply blood to the **heart** muscle and no alternative blood supply exists, so a blockage in the coronary arteries reduces the supply of blood to **heart** muscle.

**Ischaemic heart disease**

| | |
|---|---|
| **ICD-10** | I20-I25 |
| **ICD-9** | 410 (http://icd9.chrisendres.com/index.php?action=search&srchtext=410)-414 (http://icd9.chrisendres.com/index.php?action=search&srchtext=414) |

Most ischaemic **heart disease** is caused by atherosclerosis, usually present even when the artery lumens appear normal by angiography, see IVUS.

## Contents



## What is it?

- Initially there is sudden severe narrowing or closure of either the large coronary arteries and/or of coronary artery end branches by debris showering downstream in the flowing blood. It is usually felt as angina, especially if a large area is affected.
- The narrowing or closure is predominantly caused by the covering of atheromatous plaques within the wall of the artery rupturing, in turn leading to a **heart** attack (**Heart** attacks caused by just artery narrowing are rare).
- A **heart** attack causes damage to **heart** muscle by cutting off its blood supply.

This can cause:

- Temporary damage and pain (ischemia)
- Loss of muscle activity (acute **heart** failure)
- Permanent **heart** muscle damage, **heart** muscle does not grow back (acute myocardial infarction /infarct)
- Long term loss of **heart** muscle activity (chronic **heart** failure)
- Cardiac arrhythmias: irregular heartbeat which can be fatal. Most death is due to arrhythmias, usually tachyarrhythmias.
- Other structural damage to the **heart** including damaged **heart** valves, actual perforation of the **heart** and a thin walled

fibrous floppy **heart**.

# Prevention

Prevent or delay atherosclerosis.

- **Do not smoke**
- **Maintain low blood pressure** - prevent/treat hypertension (high blood pressure)
- **Exercise frequently** - exercising the **heart** muscle strengthens it, like any other
- **Avoid obesity** - increasing body fat stores, especially intra-abdominal fat, increases serum cholesterol, triglycerides, insulin requirements and promotes Diabetes Mellitus plus chronically increases **heart** muscle workload.
- **Avoid trans-fats** - these are found in any chemically modified fat product, such as margarine, in hydrogenated fats, and especially in superheated fats (such as those used for commercial deep frying). These fats are unreactive (not fitting in the enzymes designed for cis-fats) and should not be consumed in any amount; however, in many western countries, limitation may be the only practical option. Some mono-unsaturated fats are beneficial in reducing the risk of **heart disease** when consumed in moderation. When consumed in excess, however, other health concerns arise. An increase in polyunsaturated fats is also warranted in most American diets. Dietary cholesterol intake is known to have only limited effect on serum cholesterol.
- **Monitor and reduce cholesterol** - take LDLipoprotein cholesterol reducing and HDLipoprotein raising drugs and verfiy both LDLipoprotein particle counts and quantitative large HDLipoprotein response to treatment
- **Avoid shift work**
- **Eat vitamin C** - this micronutrient maintains healthy blood vessels (see scurvy), and prevents tears and fissures in the lumen wall that act as condensation nuclei on which the cholesterol molecules aglommerate

# Treatment of a heart attack.

The option required depends on the situation.

- Specialised coronary care (the sooner the better); most deaths are due to sudden onset arrhythmias - time is crucial to survival.
- Cardiopulmonary resuscitation (breathing support, pulse and BP monitoring & possible chest compressions).
- A defibrillator can stop cardiac arrhythmias.
- An artificial pacemaker can speed up cardiac bradyarrhythmias.
- Drugs such as adrenaline can increase **heart** rate and strength of contractions, although also promote tachyarrhythmias.
- Thrombolytic agents can clear away compounding blood clots.
- Anticoagulation can impede additional blood clots.
- Inotropic drugs will raise blood pressure.
- Unblock arteries with angioplasty ("balloon angioplasty with or without stents") or surgery.

# After a heart attack

- Possible angioplasty or cardiac surgery.
- Possibly the regular administration of anti-coagulants to prevent further blood clot complications.
- Possibly the administration of drugs to reduce **heart** arrhythmias although they many also induce arrhythmias.
- Lifestyle modifications are important in prevention of a second MI; increased exercise, reduction of stress, and improved dietary considerations are perhaps most important

Homoeopathic Management (after the acute symptoms have passed):

- Crategus Q for removal of plaques.
- Cactus Grandiflorus Q or 30th potency for acute pain in chest.
- Naja T 30 for breathlessness and sinking sensation.
- Stropanthus Hispidus for tonign the **heart** and its functions.
- Digitalis 30 and Calcarea Ars 30 for improving Arrythmias.

# External Link

- **Ischemic Heart Disease**

Retrieved from "http://en.wikipedia.org/wiki/Ischaemic_heart_disease"

Category: Cardiology